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=> s (feces OR fecal) AND transglutaminase  
36 FILES SEARCHED...

L1 95 (FECES OR FECAL) AND TRANSGLUTAMINASE

=> s (feces OR fecal) (p) transglutaminase  
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH  
FIELD CODE - 'AND' OPERATOR ASSUMED 'FECAL) (P) TRANSGLUT'  
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH  
FIELD CODE - 'AND' OPERATOR ASSUMED 'FECAL) (P) TRANSGLUT'



CODEN: CGHLAW; ISSN: 1542-3565

PUBLISHER: Elsevier Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Background & Aims: Undiagnosed patients with symptoms of celiac sprue often present to physicians after establishing dietary gluten exclusion. Although they must resume a gluten-containing diet for evaluation, there are no guidelines regarding duration of the gluten challenge, gluten dose, or monitoring parameters. We investigated the effects of a short-term gluten challenge in asymptomatic treated adult celiac patients on intestinal absorption and celiac antibody tests. Methods: Eight adult asymptomatic celiac patients consumed either 5 or 10 g of partially hydrolyzed gluten per day in an orange juice mixture for 21 days while maintaining their usual gluten-free diet. A symptom questionnaire, serum antibodies (antigliadin Ig [Ig]A and anti-**transglutaminase** IgA and IgG), D-xylose urine excretion test, and 72-h quant. **fecal** fat test were monitored. Results: Two patients (25%) had at least 1 abnormal celiac antibody test at baseline. There was no increase in antibodies during gluten exposure compared with baseline for any of the patients ( $P > .05$ ). At baseline, 1 patient had abnormal urine xylose excretion, and 3 patients had abnormal **fecal** fat values. At day 15 of gluten challenge, all patients had reduced xylose absorption compared with baseline ( $P = .0019$ ), and 5 of 8 participants (63%) reduced their xylose excretion to the abnormal range. Seven of 8 patients (88%) had increased **fecal** fat excretion at day 15 ( $P = .026$ ), and 6 of these (75%) had steatorrhea by day 15. Conclusions: Short-term gluten challenge in asymptomatic adult celiac patients produces carbohydrate and fat malabsorption but does not increase **transglutaminase** and antigliadin antibody titers.

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 13

MEDLINE on STN

ACCESSION NUMBER: 2005460659 MEDLINE

DOCUMENT NUMBER: PubMed ID: 16127984

TITLE: Significance of smooth muscle/anti-actin autoantibodies in celiac disease.

COMMENT: Comment in: Acta Gastroenterol Latinoam. 2005;35(2):79-82. PubMed ID: 16127983

AUTHOR: Pedreira Silvia; Sugai Emilia; Moreno Maria Laura; Vazquez Horacio; Nivèloni Sonia; Smecuol Edgardo; Mazure Roberto; Kogan Zulema; Maurino Eduardo; Bai Julio C

CORPORATE SOURCE: Small Bowel Section, Department of Medicine, Dr Carlos Bonorino Udaondo Gastroenterology Hospital, Buenos Aires, Argentina.

SOURCE: Acta gastroenterologica Latinoamericana, (2005) 35 (2) 83-93.

Journal code: 0261505. ISSN: 0300-9033.

PUB. COUNTRY: Argentina

DOCUMENT TYPE: (EVALUATION STUDIES)

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200510

ENTRY DATE: Entered STN: 20050831

Last Updated on STN: 20051027

Entered Medline: 20051026

AB BACKGROUND/AIM: Smooth muscle antibody (SMA) specific for the protein actin, a major component of the cytoskeleton of epithelial cells, is one of the most prevalent non-organ specific autoantibodies in the serum of celiac disease (CD) patients. Our aim was to explore the clinical relevance of the presence of IgA type anti-actin antibody (AAA) and SMA in a series of patients with CD. METHODS: We evaluated frozen serum samples collected at diagnosis from 92 adult patients with CD and 52 control

individuals in whom CD was excluded. Patients were re-evaluated a median time of 5 yr after treatment. IgA type AAA was detected using a modified commercial ELISA assay and IgA SMA was detected using indirect immunofluorescence on primate esophagus substrate. RESULTS: At diagnosis, samples from CD patients had significantly higher AAA values than controls ( $p < 0.00001$ ). While all active CD patients had serum AAA values over the cut-off for healthy controls, we observed a very significant reduction of these antibodies after treatment ( $p > 0.0001$ ). AAA had a highly significant correlation with both, tissue, **transglutaminase** ( $r = 0.62$ ) and antigliadin ( $r = 0.60$ ,  $p < 0.00001$ ) antibodies as well as the severity of the intestinal injury ( $p < 0.05$ ). SMA was detected in sera of 35 consecutive CD patients. At diagnosis, SMA positive patients had significantly higher values of AAA ( $p < 0.0002$ ), increased number of autoimmune disorders ( $p < 0.04$ ), delayed menarche ( $p < 0.04$ ), lower hemoglobin levels ( $p < 0.01$ ), increased **fecal** a-I antitrypsin clearance ( $p < 0.01$ ) and more severe diarrhea ( $p < 0.06$ ). We also detected a trend to more severe complications at follow-up ( $p = 0.059$ ). CONCLUSIONS: Based on our findings we suggest that the presence of increased IgA AAA serum levels is a highly sensitive marker of the disturbed architecture of intestinal epithelial cells of CD patients with a potential relevance to diagnosis and follow-up. The presence of SMA seems to define a distinct subset of CD patients with a more severe clinical outcome.

L3 ANSWER 3 OF 13 IFIPAT COPYRIGHT 2005 IFI on STN DUPLICATE 2  
 AN 10565052 IFIPAT;IFIUDB;IFICDB  
 TITLE: METHOD FOR DIAGNOSING IMMUNOLOGIC FOOD SENSITIVITY;  
 DETECTING AUTOIMMUNE AND GASTROINTESTINAL DISORDERS  
 VIA PRESENCE OF HUMAN LEUKOCYTE ANTIGEN ALLELES  
 AND/OR FAILURE TO RESPOND TO BISMUTH SUBSALICYLATE  
 TREATMENT OF MICROSCOPIC COLITIS; IMMUNOASSAY  
 INVENTOR(S): Fine; Kenneth D., Dallas, TX, US  
 PATENT ASSIGNEE(S): Unassigned  
 AGENT: STORM & HEMINGWAY, L.L.P., 8117 PRESTON RD., STE.  
 460, DALLAS, TX, 75225, US

	NUMBER	PK	DATE
PATENT INFORMATION:	US 2004072272	A1	20040415
APPLICATION INFORMATION:	US 2003-670100		20030924

	APPLN. NUMBER	DATE	GRANTED PATENT NO. OR STATUS
CONTINUATION OF:	US 2001-798557	20010302	6667160

	NUMBER	DATE
PRIORITY APPLN. INFO.:	US 2000-189668P	20000315 (Provisional)
	US 2000-224470P	20000810 (Provisional)
FAMILY INFORMATION:	US 2004072272	20040415
	US 6667160	
DOCUMENT TYPE:	Utility	
	Patent Application - First Publication	
FILE SEGMENT:	CHEMICAL APPLICATION	

NUMBER OF CLAIMS: 64

AB The invention includes novel methodology for diagnosing immunologic food or drug sensitivities. The method for diagnosing food sensitivities includes using diagnoses of other related disorders, such as microscopic colitis or other chronic immunologic/autoimmune syndromes, chronic diarrhea, irritable bowel syndrome, and hepatitis C and other hepatic diseases, Crohn's disease, alcoholism, and other idiopathic

neuropsychiatric and neurologic disorders, as indicators in the diagnosis of the food sensitivity. Additionally, failure to respond to or a relapse after treatment for microscopic colitis with bismuth subsalicylate is disclosed by the present invention as being a further indicator in the diagnosis of immunologic food sensitivity. Finally, the presence of certain HLA-DQ alleles, particularly HLA-DQ1,3; -DQ1,7; -DQ1,8; and DQ1,9, HLA-DQ1,1, and at least two subtypes of the HLA-DQ1 allele identified by molecular analysis as HLA-DQB1\*0501 and HLA-DQB1\*0602, as indicators in diagnosing immunologic food sensitivity, particularly gluten sensitivity or celiac sprue, and in diagnosing the related disease of microscopic colitis and other autoimmune disorders is also disclosed by the invention. A method for food sensitivity panel testing (for sensitivities other than gluten sensitivity) by detecting IgA antibodies in serum is also disclosed. A method for testing stool samples for the presence of particular antibodies, which is more sensitive and less invasive than prior art testing methods, is also disclosed for diagnosing immunologic food sensitivities. These methods of diagnosis may be used alone or in combination to further enhance the accuracy of diagnosis.

CLMN 64

L3 ANSWER 4 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN  
DUPLICATE 3

ACCESSION NUMBER: 2005:370988 BIOSIS

DOCUMENT NUMBER: PREV200510160826

TITLE: Anti-tissue transglutaminase antibodies in inflammatory bowel disease: new evidence.

AUTHOR(S): Di Tola, Marco; Sabbatella, Luigi; Anania, Maria Cristina; Viscido, Angelo; Caprilli, Renzo; Pica, Roberta; Paoluzi, Paolo; Picarelli, Antonio [Reprint Author]

CORPORATE SOURCE: Univ Roma La Sapienza, Policlin Umberto I, Dept Clin Sci, Viale Policlin 155, I-00161 Rome, Italy  
a.picarelli@flashnet.it

SOURCE: Clinical Chemistry and Laboratory Medicine, (2004) Vol. 42, No. 10, pp. 1092-1097.  
ISSN: 1434-6621.

DOCUMENT TYPE: Article  
General Review; (Literature Review)

LANGUAGE: English

ENTRY DATE: Entered STN: 21 Sep 2005

Last Updated on STN: 21 Sep 2005

AB Antitissue **transglutaminase**, previously held to be identical to antiendomysial antibodies in celiac sprue, has been reported in inflammatory bowel disease patients. To investigate these data further, we evaluated serum and intestinal antitissue **transglutaminase** in inflammatory bowel disease patients, with respect to the Crohns disease activity index and the integrated disease activity index. Study population comprised: 49 patients with Crohns disease and 29 patients with ulcerative colitis; 45 patients with celiac sprue and 85 autoimmune patients as disease controls; and 58 volunteers as healthy controls. Immunoglobulin A (IgA) antirecombinant human tissue **transglutaminase** and antiendomysial antibody detection in sera and fecal supernatants were performed. Adsorption of positive sera with recombinant human tissue **transglutaminase** were also performed. Marked increased antitissue **transglutaminase** concentrations were found in celiac sprue, while low positive values were also found in Crohns disease and ulcerative colitis. Antiendomysial antibodies were detectable only in celiac sprue. Antigen adsorption resulted in a significant reduction of the antitissue **transglutaminase** either in celiac sprue or inflammatory bowel disease sera. A significant correlation between antitissue **transglutaminase** and Crohns disease activity index or integrated disease activity index scores was found. Antitissue **transglutaminase** was also detectable in fecal



supernatants from inflammatory bowel disease patients. Data highlight that both circulating and intestinal antitissue transglutaminases are detectable in inflammatory bowel disease, and that they are related to disease activity. These features underline that, in addition to antitissue **transglutaminase**, an antiendomysial antibody test is necessary in the diagnostic workup of celiac sprue, especially in patients with known inflammatory bowel disease.

L3 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 4  
 ACCESSION NUMBER: 2004:884034 CAPLUS  
 DOCUMENT NUMBER: 142:54292  
 TITLE: Comparison of different salivary and fecal antibodies for the diagnosis of celiac disease  
 AUTHOR(S): Halblaub, Jeannine M. Lasheras; Renno, Joerg; Kempf, Alexander; Bartel, Jan; Schmidt-Gayk, Heinrich  
 CORPORATE SOURCE: Limbach Laboratory, Heidelberg, Germany  
 SOURCE: Clinical Laboratory (Heidelberg, Germany) (2004), 50(9+10), 551-557  
 CODEN: CLLAFP; ISSN: 1433-6510  
 PUBLISHER: Verlag Klinisches Labor  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB To investigate the detectability and expressiveness of salivary and **fecal** anti-gliadin (AGA), anti-endomysium (EMA), and anti-tissue **transglutaminase** (ATA) antibodies, 127 salivary and 160 **fecal** samples of healthy volunteers and salivary and **fecal** samples of 17 patients with histol. proven and 9 patients with suggested celiac disease were studied. With all salivary parameters and **fecal** IgA AGA, IgM AGA, IgA EMA, and IgG EMA, healthy volunteers and patients showed partially overlapping results. The most promising results in the study with higher concns. in patients with celiac disease were obtained by **fecal** scIgA AGA and a combined determination of **fecal** IgA AGA, IgG AGA, and IgM AGA. Further investigations should be performed with **fecal** IgA EMA and scIgA ATA based on human recombinant tissue **transglutaminase**. One patient with histol. proven celiac disease had normal serol. but high **fecal** scIgA AGA and scIgA ATA values. This patient emphasizes the importance of **fecal** antibody determination for the diagnosis of celiac disease, at least in patients with suggested celiac disease and neg. serum antibodies.

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 13 IFIPAT COPYRIGHT 2005 IFI on STN  
 AN 03991426 IFIPAT;IFIUDB;IFICDB  
 TITLE: METHOD FOR DIAGNOSING IMMUNOLOGIC FOOD SENSITIVITY;  
 PANEL TESTING OF FECES FOR ANTIBODIES  
 INVENTOR(S): Fine; Kenneth D., 6919 Pasadena Ave, Dallas, TX, 75214  
 PATENT ASSIGNEE(S): Unassigned  
 PRIMARY EXAMINER: Nguyen, Bao-Thuy L  
 AGENT: Barnes Robin L.  
 Storm & Hemingway, L.L.P.

	NUMBER	PK	DATE
PATENT INFORMATION:	US 6667160	B2	20031223
	US 2001036639	A1	20011101
APPLICATION INFORMATION:	US 2001-798557		20010302
EXPIRATION DATE:	2 Mar 2021		

	NUMBER	DATE
PRIORITY APPLN. INFO.:	US 2000-189668P	20000315 (Provisional)

FAMILY INFORMATION: US 2000-224470P 20000810 (Provisional)  
US 6667160 20031223  
US 2001036639 20011101  
DOCUMENT TYPE: Utility  
Granted Patent - Utility, with Pre-Grant Publication  
CERTIFICATE OF CORRECTION  
CORRECTION DATE: 13 Jul 2004  
FILE SEGMENT: CHEMICAL  
GRANTED

PARENT CASE DATA:

This application claims priority to U.S. Provisional Application Serial Nos. 60/189,668 filed on Mar. 15, 2000 and No. 60/224, 470 filed on Aug. 10, 2000.

NOTE: INDEXED FROM APPLICATION  
Subject to any Disclaimer, the term of this patent is extended or adjusted under 35 USC 154(b) by 128 days.

NUMBER OF CLAIMS: 17

AB The invention includes novel methodology for diagnosing immunologic food or drug sensitivities. A method for diagnosing food sensitivities includes using diagnoses of other related disorders as indicators in the diagnosis of the food sensitivity. Additionally, failure to respond to or a relapse after treatment for microscopic colitis with bismuth subsalicylate is disclosed as being a further indicator in the diagnosis of immunologic food sensitivity. Finally, the presence of certain HLA-DQ alleles, particularly HLA-DQ1,1; DQ1,3; -DQ1,7; -DQ1,8; and -DQ1,9 as indicators in diagnosing immunologic food sensitivity is also disclosed by the invention. A method for food sensitivity panel testing (for sensitivities other than gluten sensitivity) by detecting IgA antibodies in serum is also disclosed. A method for testing stool samples for the presence of particular antibodies is also disclosed for diagnosing immunologic food sensitivities. These methods of diagnosis may be used alone or in combination to further enhance accuracy of diagnosis.

NTE INDEXED FROM APPLICATION  
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ACCESSION NUMBER: 2004-0100299 PASCAL

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TITLE (IN ENGLISH): How often is a positive faecal occult blood test the result of coeliac disease?

AUTHOR: LOGAN Richard F. A.; HOWARTH Georgina F.; WEST Joe; SHEPHERD Kate; ROBINSON Michael H. E.; HARDCASTLE Jack D.

CORPORATE SOURCE: Department of Epidemiology and Public Health, University Hospital, University of Nottingham, Nottingham, United Kingdom; Department of Surgery, University Hospital, University of Nottingham, Nottingham, United Kingdom

SOURCE: European journal of gastroenterology & hepatology, (2003), 15(10), 1097-1100, 11 refs.

ISSN: 0954-691X

DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: United States

LANGUAGE: English

AVAILABILITY: INIST-22107, 354000113365680060

AN 2004-0100299 PASCAL

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AB Background and aims It has been reported that occult gastrointestinal bleeding as detected by faecal occult blood (FOB) testing can occur in coeliac disease. This study examines whether a positive FOB is a feature of coeliac disease and whether FOB-positive subjects need investigation for coeliac disease. Methods First, the records of patients on the Nottingham Register for Coeliac Disease were reviewed for positive FOB testing. Second, the Nottingham colorectal cancer screening trial database was also reviewed to examine how many coeliac patients on the Register had participated and to examine their FOB results. Finally, sera from 309 screening trial participants who were FOB-positive but had no colonic abnormality were screened for immunoglobulin A (IgA) gliadin and IgA endomysial and human tissue **transglutaminase** (tTG) IgA antibodies. Results Five of 590 patients on the Register had had FOB tests at the time of diagnosis; four had positive tests during investigation of diarrhoea and/or anaemia. Of 21 patients on the Register who had participated in the colorectal cancer screening trial, one had a positive FOB test and was found to have a rectal tubulo-villous adenoma. Of the 309 FOB-positive patients, 7% (22 subjects) were positive for IgA gliadin antibodies, but none had IgA endomysial antibodies detected and two subjects had positive human tTG antibody assays for coeliac disease. Conclusions Occult gastrointestinal bleeding occurs in a small number of symptomatic coeliac disease patients before diagnosis, but is no more frequent in treated and undetected coeliac disease patients than in the general population. Unless there are other indications, coeliac disease does not need to be considered in the investigation of a positive FOB test.

L3 ANSWER 8 OF 13 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:530587 SCISEARCH

THE GENUINE ARTICLE: 688TD

TITLE: Guidelines for the investigation of chronic diarrhoea, 2nd edition

AUTHOR: Thomas P D; Forbes A (Reprint); Green J; Howdle P; Long R; Playford R; Sheridan M; Stevens R; Valori R; Walters J; Addison G M; Hill P; Brydon G

CORPORATE SOURCE: St Marks Hosp, Dept Gastroenterol, Harrow HA1 3UJ, Middx, England (Reprint); Musgrave Pk Hosp, Dept Gastroenterol, Taunton, Somerset, England; Llandough Hosp, Dept Gastroenterol, Penarth, S Glam, Wales; St Jamess Univ Hosp, Dept Gastroenterol, Leeds, W Yorkshire, England; City Hosp, Dept Gastroenterol, Nottingham NG5 1PB, England; Hammersmith Hosp, Dept Gastroenterol, London, England; St James Hosp, Dept Radiol, Leeds, W Yorkshire, England; E Oxford Hlth Ctr, GP, Oxford, England; Gloucestershire Royal Hosp, Dept Gastroenterol, Gloucester GL1 3NN, England; Royal Manchester Childrens Hosp, Dept Clin Chem, Manchester M27 1HA, Lancs, England; Hope Hosp, Dept Clin Chem, Manchester, Lancs, England; Western Gen Hosp, Edinburgh EH4 2XU, Midlothian, Scotland

COUNTRY OF AUTHOR: England; Wales; Scotland

SOURCE: GUT, (JUL 2003) Vol. 52, Supp. [5], pp. 1-15.  
ISSN: 0017-5749.

PUBLISHER: B M J PUBLISHING GROUP, BRITISH MED ASSOC HOUSE, TAVISTOCK SQUARE, LONDON WC1H 9JR, ENGLAND.

DOCUMENT TYPE: General Review; Journal

LANGUAGE: English

REFERENCE COUNT: 213

ENTRY DATE: Entered STN: 13 Jul 2003

Last Updated on STN: 13 Jul 2003

L3 ANSWER 9 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

DUPLICATE 5

ACCESSION NUMBER: 2002:168343 BIOSIS  
 DOCUMENT NUMBER: PREV200200168343  
 TITLE: Antiendomysial antibody detection in fecal supernatants: In vivo proof that small bowel mucosa is the site of antiendomysial antibody production.  
 AUTHOR(S): Picarelli, Antonio [Reprint author]; Sabbatella, Luigi; Di Tola, Marco; Di Cello, Teresa; Vetrano, Stefania; Anania, Maria Cristina  
 CORPORATE SOURCE: Department of Clinical Sciences, Universita di Roma "La Sapienza", Viale del Policlinico 155, 00161, Rome, Italy  
 SOURCE: American Journal of Gastroenterology, (January, 2002) Vol. 97, No. 1, pp. 95-98. print.  
 CODEN: AJGAAR. ISSN: 0002-9270.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 5 Mar 2002  
 Last Updated on STN: 5 Mar 2002

AB OBJECTIVES: Serum antiendomysial antibodies (EMAs), highly sensitive and specific serological markers of celiac disease (CD), are detectable in culture media of biopsy samples from CD patients. This finding can be considered an in vitro evidence that intestinal mucosa is a site of EMA production. To confirm this finding, we investigated the presence of EMAs and of anti-tissue **transglutaminase** (anti-tTG), recently identified as the autoantigen of the EMA, in **fecal** supernatants of CD patients. METHODS: Twenty-one newly diagnosed CD patients, 10 treated CD patients on a gluten-free diet, and 14 control disease patients on a gluten-containing diet were enrolled. Twenty-four-hour stool collections and **fecal** supernatants were obtained from all patients in the study. Biopsy cultures were also performed. IgA EMAs were detected in sera, culture media, and **fecal** supernatants. IgA, IgG, IgM, and IgE anti-gliadin antibodies (AGAs) and IgA anti-tTG antibodies were measured in **fecal** supernatants. The weights, water content, and pHs of the 24-h stool collections were also measured. RESULTS: In all untreated CD patients EMAs were detectable in sera, culture media, and **fecal** supernatants. In treated CD patients, EMAs were detected only in culture media after in vitro gliadin challenge. No EMAs were detected in controls. Anti-tTG levels were higher in untreated CD patients than in treated CD patients and controls. IgA AGA levels were higher in untreated CD patients than in treated CD and control patients, whereas IgM AGAs were higher in both untreated and treated CD patients than in controls. No statistically significant differences were observed for IgG and IgE AGAs among the above-mentioned populations. **Fecal** weights, water content, and pHs were higher in untreated CD than in control patients. CONCLUSIONS: The presence of EMAs in **fecal** supernatants represents the in vivo proof that intestinal mucosa is a site of EMA production. Furthermore, EMA detection in the stools could be a simple and useful additional tool to clarify diagnosis in the patchy conditions of CD.

L3 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 6

ACCESSION NUMBER: 2001:693659 CAPLUS  
 DOCUMENT NUMBER: 135:240909  
 TITLE: Method for diagnosing immunologic food sensitivity  
 INVENTOR(S): Fine, Kenneth D.  
 PATENT ASSIGNEE(S): USA  
 SOURCE: PCT Int. Appl., 37 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001069251	A2	20010920	WO 2001-US7908	20010313
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2001036639	A1	20011101	US 2001-798557	20010302
US 6667160	B2	20031223		
CA 2400968	AA	20010920	CA 2001-2400968	20010313
EP 1322956	A2	20030702	EP 2001-916588	20010313
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
US 2004072272	A1	20040415	US 2003-670100	20030924
PRIORITY APPLN. INFO.:				
			US 2000-189668P	P 20000315
			US 2000-224470P	P 20000810
			US 2001-798557	A 20010302
			WO 2001-US7908	W 20010313

AB The invention includes novel methodol. for diagnosing immunol. food or drug sensitivities. The method for diagnosing food sensitivities includes using diagnoses of other related disorders, such as microscopic colitis or other chronic immunol./autoimmune syndromes, chronic diarrhea, irritable bowel syndrome, and hepatitis C and other hepatic diseases, Crohn's disease, alcoholism, and other idiopathic neuropsychiatric and neurol. disorders, as indicators in the diagnosis of the food sensitivity. Addnl., failure to respond to or a relapse after treatment for microscopic colitis with bismuth subsalicylate is disclosed by the present invention as being a further indicator in the diagnosis of immunol. food sensitivity. Finally, the presence of certain HLA-DQ alleles, particularly HLA-DQ1,3; -DQ1,7; -DQ1,8; and -DQ1,9, HLA-DQ1,1, and at least two subtypes of the HLA-DQ1 allele identified by mol. anal. as HLA-DQB10501 and HLA-DQB10602, as indicators in diagnosing immunol. food sensitivity, particularly gluten sensitivity or celiac sprue, and in diagnosing the related disease of microscopic colitis and other autoimmune disorders is also disclosed by the invention. A method for food sensitivity panel testing (for sensitivities other than gluten sensitivity) by detecting IgA antibodies in serum is also disclosed. A method for testing stool samples for the presence of particular antibodies, which is more sensitive and less invasive than prior art testing methods, is also disclosed for diagnosing immunol. food sensitivities. These methods of diagnosis may be used alone or in combination to further enhance the accuracy of diagnosis.

L3 ANSWER 11 OF 13 IFIPAT COPYRIGHT 2005 IFI on STN DUPLICATE 7  
AN 10036622 IFIPAT;IFIUDB;IFICDB  
TITLE: METHOD FOR DIAGNOSING IMMUNOLOGIC FOOD SENSITIVITY;  
PANEL TESTING OF FECES FOR ANTIBODIES  
INVENTOR(S): Fine; Kenneth D., Dallas, TX, US  
PATENT ASSIGNEE(S): Unassigned  
AGENT: CARR & STORM, L.L.P., 900 JACKSON STREET, 670  
FOUNDERS SQUARE, DALLAS, TX, 75202, US

	NUMBER	PK	DATE
PATENT INFORMATION:	US 2001036639	A1	20011101
APPLICATION INFORMATION:	US 2001-798557		20010302

NUMBER	DATE
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PRIORITY APPLN. INFO.:	US 2000-189668P	20000315 (Provisional)
	US 2000-224470P	20000810 (Provisional)
FAMILY INFORMATION:	US 2001036639	20011101
	US 6667160	20031223
DOCUMENT TYPE:	Utility	
	Patent Application - First Publication	
FILE SEGMENT:	CHEMICAL	
	APPLICATION	

NUMBER OF CLAIMS: 64

AB The invention includes novel methodology for diagnosing immunologic food or drug sensitivities. A method for diagnosing food sensitivities includes using diagnoses of other related disorders as indicators in the diagnosis of the food sensitivity. Additionally, failure to respond to or a relapse after treatment for microscopic colitis with bismuth subsalicylate is disclosed as being a further indicator in the diagnosis of immunologic food sensitivity. Finally, the presence of certain HLA-DQ alleles, particularly HLA-DQ1,1; DQ1,3; -DQ1,7; -DQ1,8; and -DQ1,9 as indicators in diagnosing immunologic food sensitivity is also disclosed by the invention. A method for food sensitivity panel testing (for sensitivities other than gluten sensitivity) by detecting IgA antibodies in serum is also disclosed. A method for testing stool samples for the presence of particular antibodies is also disclosed for diagnosing immunologic food sensitivities. These methods of diagnosis may be used alone or in combination to further enhance accuracy of diagnosis.

CLMN 64

L3 ANSWER 12 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on  
STN DUPLICATE 8

ACCESSION NUMBER: 2002:96321 BIOSIS  
DOCUMENT NUMBER: PREV200200096321  
TITLE: Diagnosis of celiac sprue.  
AUTHOR(S): Farrell, Richard J. [Reprint author]; Kelly, Ciaran P.  
CORPORATE SOURCE: Gastroenterology Division, Beth Israel Deaconess Medical Center, 330 Brookline Avenue, Dana 501, Boston, MA, 02215, USA  
SOURCE: American Journal of Gastroenterology, (December, 2001) Vol. 96, No. 12, pp. 3237-3246. print.  
CODEN: AJGAAR. ISSN: 0002-9270.  
DOCUMENT TYPE: Article  
General Review; (Literature Review)  
LANGUAGE: English  
ENTRY DATE: Entered STN: 24 Jan 2002  
Last Updated on STN: 25 Feb 2002

AB Celiac sprue is a common lifelong disorder affecting 0.3-1% of the Western world and causing considerable ill health and increased mortality, particularly from lymphoma and other malignancies. Although high prevalence rates have been reported in Western Europe, celiac sprue remains a rare diagnosis in North America. Whether celiac sprue is truly rare among North Americans or is simply underdiagnosed is unclear, although serological screening of healthy American blood donors suggests that a large number of American celiacs go undiagnosed. Celiac sprue is an elusive diagnosis, and often its only clue is the presence of iron or folate deficiency anemia or extraintestinal manifestations, such as osteoporosis, infertility, and neurological disturbances. The challenge for gastroenterologists and other physicians is to identify the large population of undiagnosed patients that probably exists in the community and offer them treatment with a gluten-free diet that will restore the great majority to full health and prevent the development of complications. The advent of highly sensitive and specific antiendomysium and tissue **transglutaminase** serological tests has modified our current approach to diagnosis and made **fecal** fat and D-xylose

absorption testing obsolete. A single small bowel biopsy that demonstrates histological findings compatible with celiac sprue followed by a favorable clinical and serological response to gluten-free diet is now considered sufficient to definitely confirm the diagnosis. We review the wide spectrum of celiac sprue, its variable clinical manifestations, and the current approach to diagnosis.

L3 ANSWER 13 OF 13 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on  
STN DUPLICATE 9

ACCESSION NUMBER: 1996:528134 BIOSIS  
DOCUMENT NUMBER: PREV199699250490  
TITLE: The epsilon-(gamma-glutamyl)lysine moiety in crosslinked casein is an available source of lysine for rats.  
AUTHOR(S): Seguro, Katsuya; Kumazawa, Yoshiyuki; Kuraishi, Chiya; Sakamoto, Hiroko; Motoki, Masao  
CORPORATE SOURCE: Ajinomoto Co. Inc., 1-1 Suzuki-cho, Kawasaki-ku, Kawasaki-shi 210, Japan  
SOURCE: Journal of Nutrition, (1996) Vol. 126, No. 10, pp. 2557-2562.  
CODEN: JONUAI. ISSN: 0022-3166.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
ENTRY DATE: Entered STN: 22 Nov 1996  
Last Updated on STN: 23 Nov 1996

AB To determine bioavailability, expressed as the protein efficiency ratio (PER) and biological value (BV) in rats, of the epsilon-(gamma-glutamyl)lysine (epsilon-(gamma-Glu)Lys) moiety in crosslinked proteins, we prepared heavily crosslinked (21.5 mu-mol epsilon-(gamma-Glu)Lys/g casein) and intermediately crosslinked (13.6 mu-mol epsilon-(gamma-Glu)Lys/g casein) casein, using microbial **transglutaminase**. In Experiment 1, rats were assigned to one of four diets (heavily or intermediately crosslinked caseins, intact casein or non-protein diet) for 4 wk to evaluate the bioavailability of the epsilon-(gamma-Glu)Lys moiety in crosslinked casein as the sole source of dietary protein. Rats that were fed intact casein and the two crosslinked caseins had similar growth rates, PER, and BV, indicating that crosslinked caseins supported the growth of rats similarly to the intact casein. In Experiment 2, heavily crosslinked casein was added to wheat gluten-based diets in concentrations of 20 and 40 g/kg diet to evaluate the bioavailability of lysine in the epsilon-(gamma-Glu)Lys moiety of the casein as a lysine supplement for lysine-poor gluten. One of six diets (heavily crosslinked or intact casein diets in the two concentrations, gluten diet, or non-protein diet) was fed to rats for 4 wk. No significant differences in food intake, body weight gain, PER or BV were observed among rats fed the intact or crosslinked casein diets at either 2 or 4 g/100 g casein. These results suggest that the epsilon-(gamma-Glu)Lys moiety in crosslinked caseins are absorbed and therefore supplement the gluten. HPLC analysis of urine and **feces** of rats fed the crosslinked caseins actually confirmed that approx 99% of the epsilon-(gamma-Glu)Lys moiety was absorbed in the body.

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